Treatment of Trapped CCF by Direct Puncture of the Cavernous Sinus by Infraocular Trans-SOF Approach

Case Report and Anatomical Basis

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Summary

We present a case of recurrent carotid-cavernous fistula after prior ipsilateral carotid artery ligation. Due to lack of endovascular access, embolization was performed by direct puncture of the cavernous sinus via a transorbital approach. Operative technique and an anatomical basis for treatment are described.

Introduction

Shear forces at points of carotid artery fixation, as it enters and leaves the cavernous sinus, during acceleration-deceleration injury can lead to development of a traumatic carotid-cavernous fistula (CCF). Most commonly the CCF results from a single tear in the cavernous carotid artery that fistulizes with the cavernous sinus. The CCF causes increased venous pressure in the cavernous sinus and outflow veins, including the superior and inferior ophthalmic veins. This leads to venous engorgement and signs of pulsating exophthalmos, conjunctival chemosis, motility restriction, ocular ischemia and vision loss. Subarachnoid haemorrhage may result if venous drainage is primarily through cortical veins or brainstem edema and cranial neuropathies if venous drainage is through the basilar plexus [1].

Historically, treatment schemes included carotid ligation in various combinations, surgical trapping by ligating the proximal internal carotid and clipping the supraclinoid carotid artery, or embolizing the fistula with muscle [2]. These techniques were fraught with unsatisfactory results from high morbidity, incomplete or no symptom relief, and delayed recurrence of the CCF because the fistula itself was not occluded. These techniques did not address the numerous external to cavernous carotid collateral pathways that can reconstitute the trapped cavernous segment and cause the CCF to recur. Current standard therapy is transarterial occlusion of the fistula [3]. Transvenous approaches including the inferior petrosal sinus, superior orbital vein or cortical veins have been used when trans-arterial access is not anatomically feasible [4]. We describe a technique and anatomical basis for treatment of a CCF by direct puncture of the cavernous sinus via a trans-orbital approach.

Case Report

A 35-year-old right-handed male presented with sudden onset severe headache and nuchal rigidity. The neurological examination was normal and there were no stigmata of ocular ve-

nous hypertension. The history was significant for a left CCF resulting from a gunshot wound to the head at age nine. The CCF was treated by trapping of the carotid artery with placement of an aneurysm clip across the supraclinoid carotid artery and ligation of the cervical internal carotid artery.

Head CT demonstrated a small, left cerebellar hemisphere parenchymal haematoma and subarachnoid haemorrhage predominantly layering within cerebellar fissures and sulci. Cerebral angiography revealed a ligated left internal carotid artery and reconstitution of the cavernous internal carotid artery and through external carotid vessels (figure 1). A single hole carotid-cavernous fistula resulted in early opacification of the left cavernous sinus with venous drainage to posterior fossa tentorial and cortical veins and the basal vein of Rosenthal. There was no opacification of the superior ophthalmic vein, contralateral cavernous sinus, or inferior petrosal sinus.

Full informed consent was obtained for embolization of the cavernous sinus using either a transvenous or a direct puncture technique. After general anesthesia had been induced, attempts to traverse the thrombosed inferior petrosal sinus using a transvenous, transfemoral route were unsuccessful. Therefore, in the absence of direct transarterial or transvenous access to the cavernous sinus, direct puncture was performed. Through a small dermatotomy in the skin along the inferolateral aspect of the left eyelid, a 16-gauge angiocath was advanced posterior and inferiorly to avoid injury to the globe. Using fluoroscopic guidance and roadmap angiographic technique, the angiocath was directed along the inferolateral wall of the orbit, toward the superior orbital fissure (SOF) (figure 2A,B). It was then advanced through the SOF into the cavernous sinus and position was confirmed with contrast injection (figure 2C). A 4 F vertebral catheter (Cordis, Miami, FL) was used to canulate the left common carotid artery for initial diagnostic cerebral angiography and subsequent control angiography during embolization.

Embolization was performed with 0.038 in Gianturco coils (Cook, Bloomington, IN) (figure 3A) deployed via the 16 g angiocath in the cavernous sinus (eight 8 x 10 mm, four 5 x 5, and eight 4 x 3 mm coils). A 4 F catheter was connected to the angiocatheter using a touyborst adapter to allow ergonomic deployment of coils and to increase distance between the operator and image intensifier. Post-embolization angiography (figure 3B) showed no opacification of the cavernous sinus with obliteration of the CCF. The angiocath was removed and the patient was transferred to the neuro-intensive care unit for observation.

Initial postoperative exam revealed mild left periorbital swelling, proptosis, chemosis and ptosis. These symptoms improved during the hospital admission and at discharge (post-op day 6) there was only mild chemosis present. Although the patient developed no signs of vasospasm and serial transcranial Doppler exams were normal, prophylactic treatment with nimodipine and hemodilution, hypertensive and hypervolemic therapies were instituted. Follow-up angiography at 6 months showed obliteration of the CCF.

Discussion

Anatomical challenges complicating the endovascular management of CCF:

This case demonstrates the problems in treating carotid-cavernous fistulae by ligating feeding arteries. Treatment of fistulae ideally should include occlusion at the site of fistulization. Surgical ligation strategies allow external carotid to internal carotid collateral arteries to reconstitute the trapped segment of the cavernous carotid artery and supply the fistula. Endovascular therapies are aimed at gaining access to the cavernous sinus and embolizing it, disallowing communication between the fistulous site in the carotid artery and cavernous sinus outflow veins, while preserving patency of the ICA whenever possible.

In our patient, venopathy secondary to long standing high flow and increased pressure likely resulted in occlusion of the superior and inferior ophthalmic veins and inferior and superior petrosal sinuses, leaving the primary venous outflow through the cortical veins to the posterior fossa. The anatomy of the CCF and venous outflow correlate with the CT findings of SAH layering in fissures and sulci of the left cerebellar hemisphere. The cavernous sinus has multiple sites of inflow and outflow including the superior ophthalmic vein, inferior ophthalmic vein, pterygoid plexus, inferior petrosal sinus, superior petrosal sinus, sphenoparietal sinus

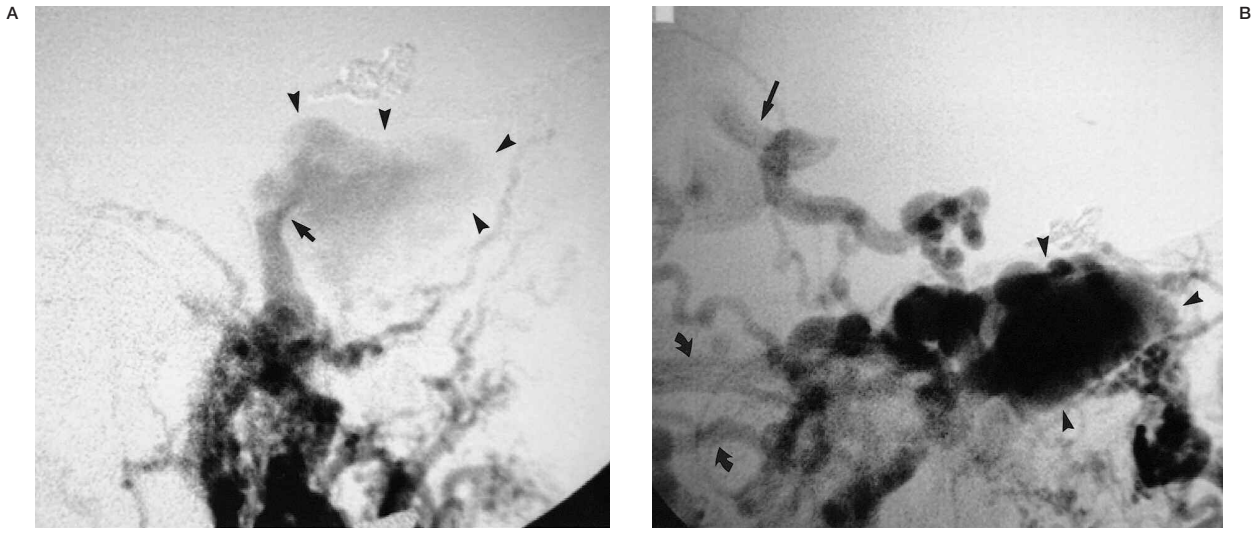

Figure 1 A) Left common carotid artery angiogram, early arterial phase, lateral projection. Angiogram demonstrates reconstitution of the cavernous carotid artery with a single hole fistula (black arrow) opacifying the cavernous sinus (arrowheads). B) Left common carotid artery angiogram, venous phase, lateral projection. The cavernous sinus is opacified (arrowheads) with venous outflow through the basal vein of Rosenthal and vein of Galen (black arrow), tentorial and cerebellar veins (curved arrows).

and temporal veins. Venous flow patterns determine patient symptoms, risk and potential routes of treatment. Commonly, venous drainage from CCF is through the ophthalmic veins, which result in ptosis, chemosis and proptosis. However, if venous drainage is primarily via the IPS or cortical veins, the patient may not have symptoms referable to the eye. Cortical venous drainage is associated with high risk for intracranial haemorrhage and poor outcome and is an indication for urgent therapy [1,5].

In our case, access to the fistula was limited due to prior ligation of the cervical internal and supraclinoid carotid arteries and pathologic occlusion of the commonly used transvenous routes (superior ophthalmic vein and inferior petrosal sinus). Standard therapy for direct CCF is transarterial occlusion with detachable balloons [3]. Transvenous routes are used when transarterial balloon occlusion attempts fail or there is carotid occlusion either from trauma or a prior surgical trapping procedure [4]. Transvenous embolization of the cavernous sinus has been described using inferior petrosal sinus, superior ophthalmic vein, pterygoid plexus and cortical vein access [4,6,7]. Without a transarterial or transvenous route available, the options for access to the cavernous sinus were either a percutaneous transorbital approach or craniotomy and exposure of the cavernous sinus or cortical vein. We chose a transorbital, superior orbital fissure approach because it is less invasive than open craniotomy and could be performed in the radiology department where high resolution, biplane angiography is available.

The transorbital, superior orbital fissure approach to the cavernous sinus has been previously described by Teng et Al, first in a case report, then in a case series with successful embolization in 11 patients with direct CCF [8,9]. Ten of their patients had been initially treated with carotid ligation or balloon occlusion of the proximal ICA. Transient post-operative ptosis was the only reported post-operative complication, occurring in two patients. However, subarachnoid haemorrhage due to transgression of the subarachnoid space, vision loss due to retrobulbar haematoma or direct injury of the optic nerve or ophthalmic artery are potential complications. Safe transorbital needle placement into the cavernous sinus necessitates knowledge of the anatomical relationships between the CS, SOF, IOF and their contents.

Anatomical Considerations for Transorbital Access to the Cavernous Sinus

The cavernous sinus (CS) is a large, parasellar, venous sinusoid posterior to the orbital apex. It envelops the cavernous carotid artery

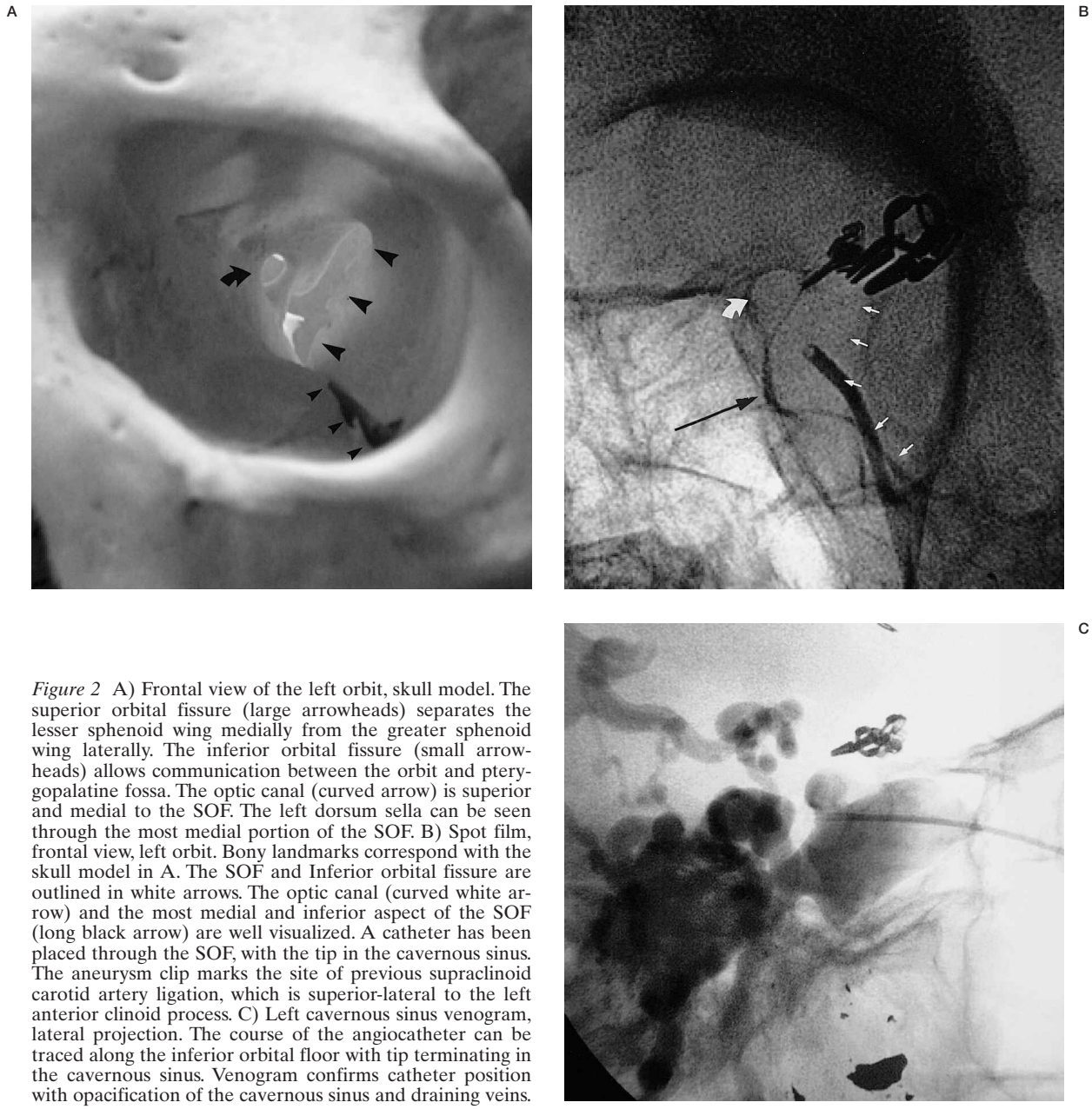

Figure 2 A) Frontal view of the left orbit, skull model. The superior orbital fissure (large arrowheads) separates the lesser sphenoid wing medially from the greater sphenoid wing laterally. The inferior orbital fissure (small arrowheads) allows communication between the orbit and pterygopalatine fossa. The optic canal (curved arrow) is superior and medial to the SOF. The left dorsum sella can be seen through the most medial portion of the SOF. B) Spot film, frontal view, left orbit. Bony landmarks correspond with the skull model in A. The SOF and Inferior orbital fissure are outlined in white arrows. The optic canal (curved white arrow) and the most medial and inferior aspect of the SOF (long black arrow) are well visualized. A catheter has been placed through the SOF, with the tip in the cavernous sinus. The aneurysm clip marks the site of previous supraclinoid carotid artery ligation, which is superior-lateral to the left anterior clinoid process. C) Left cavernous sinus venogram, lateral projection. The course of the angiocatheter can be traced along the inferior orbital floor with tip terminating in the cavernous sinus. Venogram confirms catheter position with opacification of the cavernous sinus and draining veins.

and CN III, IV, VI, V1, and V2. The lateral and superior margins are bordered by dura mater, which separate the CS from the subarachnoid space. The superior boundary extends posterior from the anterior clinoid process to the dorsum sellae. The inferior and medial borders are the sphenoid bone. The carotid artery enters the cavernous sinus posterior and inferiorly as it crosses the foramen lacerum and exits at the anterior clinoid process as it enters the subarachnoid space. The infero-medial portion of the SOF is anterior to the cavernous sinus.

The superior orbital fissure is a slit-like opening between the lesser sphenoid wing superioromedially and greater sphenoid wing inferolaterally. Cranial nerves (CN) III, IV, VI and the ophthalmic branch of V, lacrimal and orbital branch of the middle meningeal artery

pass through the SOF. The ophthalmic artery and optic nerve pass through the optic foramen, which is superior to the SOF. The central retinal, ethmoidal, lacrimal, muscular, and ciliary arteries are branches of the ophthalmic artery, which is the main arterial supply of the orbit. A lacrimal artery variant may originate from the middle meningeal artery and enter the orbit through the foramen of Hyrtl, lateral to the SOF [10]. The annulus of Zinn is at the orbital apex and is the site of tendinous insertion of the extra ocular muscles. The annulus of Zinn is centered between the SOF and optic foramen. Cranial nerves II, III, VI, V1 and the ophthalmic artery course through the annulus of Zinn, while CN IV passes medially.

When directing the needle through the SOF into the CS, it is important to enter the cavernous sinus anteriorly to avoid transgression of the subarachnoid space. This can be done by angling the image intensifier in an oblique projection, oriented to the long axis of the orbit so that the extreme medial portion of the SOF is visible and used as a target (figure 2). Biplane "road-map" angiogram should be performed to opacify the CS and confirm its borders. However, the subarachnoid space is not opacified and avoiding it requires careful study of bony landmarks. The course of the needle in the orbit is extraconal anteriorly, but probably becomes intraconal posteriorly, piercing the intermuscular septum. The optic nerve is centered within the intraconal space and the needle tip should be kept inferior to the optic canal. Cranial nerve IV, V1, V2 branches, superior division of III and the SOV enter the orbit through the superior and lateral portion of the SOF. Cranial nerve VI, inferior division of III course inferior to the optic nerve and ophthalmic artery and are positioned medially in the SOF [11]. Although no cranial neuropathies or orbital haematoma has been described by Teng et Al or in our case, despite accurate needle placement, injury to these structures is possible [9].

The cavernous carotid artery may be visible as a filling defect within the cavernous sinus during venogram through the angiocatheter. If the cavernous carotid artery has been surgically trapped, the artery may be punctured with the angiocatheter and embolized. This technique allows embolization of the fistula with fewer coils than when embolizing the cavernous sinus [9].

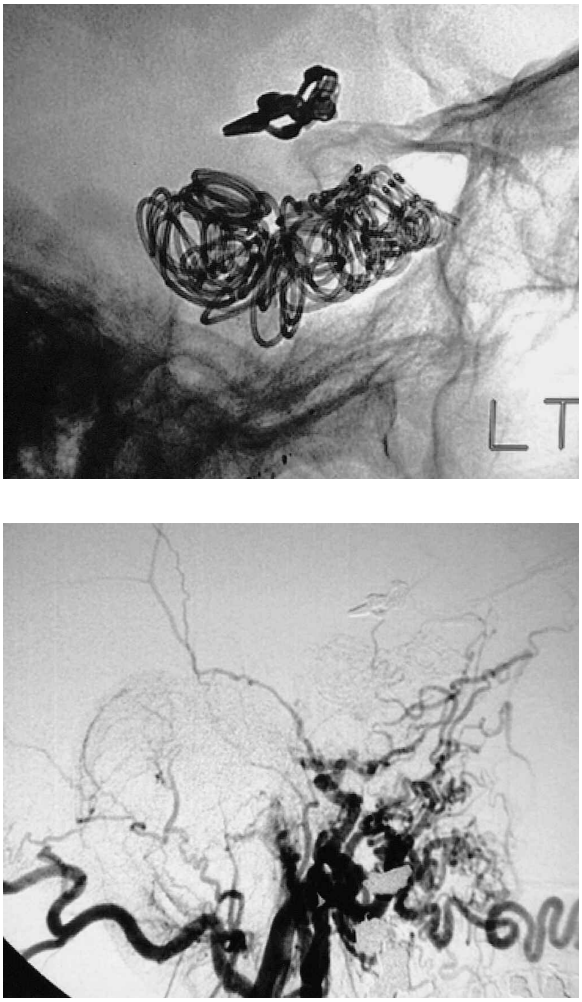

Figure 3 A) Spot film of orbit and skull, lateral view. Gianturco coils fill the left cavernous sinus. B) Left common carotid artery angiogram, lateral projection. Angiogram shows no opacifiation of the cavernous sinus or cavernous carotid artery. Due to lack of shunting, there is improved opacification of the external carotid artery branches.

Device Considerations

Once the cavernous sinus has been accessed, a variety of materials are available for embolization. Detachable balloons require a 7 F system, which significantly increases the diameter or the access catheter and would increase risk of injuring transgressing cranial nerves and arteries. Liquid embolic agents, such as NBCA (N-butyl-2-cyanoacrylate)(Cordis, Miami, FL) glue, are less controlled than coils and may require giving up access to prevent gluing the catheter to the CS. Nevertheless, glue may be a useful adjunct embolization material in some

cases [9]. Detachable or pushable coils allow controlled, stepwise embolization that can be performed with small catheters and with preservation of access to the CS. We chose stainless steel, fibered coils because the patient was already MRI incompatible, deployment can be done through a 16 g angiocatheter, they are highly thrombogenic and with prior supraclinoid carotid artery clipping, there was no risk of the coils exiting the fistula and embolizing in a distal arterial branch.

With careful study of relevant anatomy and fluoroscopic bony landmarks, the transorbital, SOF approach is feasible and is a viable alternative when other methods to gain access to the cavernous sinus fail.

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